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DESIGN CRITERIA FOR A PREDICTIVE ECOLOGICAL EFFECTS MODELING SYSTEM

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Abstract

The capability to predict the environmental fate of chemicals based on their chemical properties is well developed and widely practiced. Poorly developed, however, is the capability to predict the effects of those chemicals. To address this deficiency, significant effort has been directed to the creation of mathematical models to predict the effects of toxicants in aquatic systems. These models consist of two parts: an ecological component and a toxicological component. The ecological components are organized at levels of integration and resolution intended to be relevant to the kinds of expected problems and questions to be addressed in determining whether to permit or prohibit the use of a new chemical. Models of systems in which the organisms are assumed to have reached steady state with respect to the level of toxicant are at the coarsest level of resolution. Populations are represented at an intermediate and ecosystems at a fine scale of resolution. A toxicological model in corresponding detail is associated with each level to provide predictions of effects. This model assumes for all levels that a common threshold concentration exists at which the organism dies. With this assumption, a model for exchange with the environment, and assumptions of rapid internal distribution of the toxicant, the time to death for an individual depends on the fraction of its body comprised of fat. Probability of death by the end of a fixed exposure time is a function of the statistical distribution of the fraction of fat in the organisms' bodies.

Introduction

Regulation of new chemicals is made difficult by a lack of comprehensive data describing the properties of the compounds that determine their environmental behavior. Many of the activities involved in providing the background information for regulatory decisions, therefore, are predictive. These activities include calculating the physical and chemical properties of the new compound; making inferences about the compound's environmental behavior (movement and reactions) and about its levels in various environments in which organisms are exposed; and predicting the kinds of effects, their intensity, and the manner in which they are propagated in an ecosystem. The capability to carry out these activities varies considerably, and the way that each activity should be carried out (e.g., what specific endpoints should be predicted) is yet to be fully defined.

In this paper, design criteria for a comprehensive ecological effects modeling system are discussed. Many of the concepts discussed here are well developed (e.g., the toxicological model for neutral hydrophobic chemicals) whereas others (e.g., the specific implementation for the ecological model) are less well developed. Selection of measurable endpoints is discussed and desirable features of a computer implementation of these models are given.

Major Components of a Risk Analysis Modeling System

Generally, it is considered that analysis of risks to natural populations posed by the introduction of new chemicals into the environment consists of two components: determination of the fate of the toxicant and evaluation of its effects on the natural populations. These two types of analyses are often referred to as exposure and effects analysis, respectively.

Exposure analysis is directed toward producing expected concentration distributions of a chemical in the environments of interest. These values are

obtained either at steady-state conditions for chemicals that are loaded continuously into the environment at approximately constant levels, or as a function of time for chemicals that are introduced in a time-dependent manner.

Under currently available technology, it is considered that the fate of the chemical is not affected by response of any decomposer population or other process to the chemical itself. This has been found to be a good assumption, in general, although effects on microbial populations are well known. In current exposure analysis technology limitation to this assumption is necessitated by restriction of the state variables to the toxicants themselves, with the chemical and biological components of the natural environment being represented parametrically.

The major advance in exposure analysis has been the incorporation of modeling techniques that permit analyses based on chemical and environmental properties, with less reliance on empirical knowledge of the behavior of the chemical in natural systems. This approach is taken by EXAMS (1) and other models incorporating descriptions of equilibrium and kinetic processes that depend on measured or estimated values for properties of the chemical and on selected properties of the environment. This approach has enabled predictive analysis, i.e., the analysis of the fate of chemicals whose environmental behavior has not been observed.

Effects analysis is directed toward identifying and quantifying expected responses in biotic populations resulting from toxicant exposure. Currently, no predictive effects analysis systems exist. Analysis of effects in ecological systems is usually accomplished by extension of laboratory results obtained using bioassay or similar techniques. These techniques will continue to be useful, but other approaches are needed to provide a rational foundation for predicting effects in organisms that have not been tested, for predicting

indirect effects, and for providing a theoretical basis for the relationship of the expected response difference between laboratory and field populations, and between acute and chronic effects. A predictive ecological effects analysis tool would necessarily be based on descriptions of physiological processes, population characteristics, and ecological processes, and on values for properties of the chemical and the exposed organisms. Such a capability would complement existing exposure analysis techniques, thereby providing a powerful ecological risk analysis capability.

The use of models for analysis of exposure, effects, and overall risk is not likely to produce final evidence or grounds on which to base regulatory decisions. Instead models are used to supplement the capability of analysts, and therefore, are useful only in a "heuristic mode." This point of view has been discussed by Overton and Lassiter (2) for the use of exposure models and applies equally strongly to the use of models for risk analysis. The term "heuristic mode" is used to denote the suggestive and comprehension-promoting value of results obtained from models used in this manner. Comprehension of response of a system to chemicals to the extent that the appropriate questions are raised, relevant data are obtained, and sound conclusions are drawn, is what should be required of a model. Design and evolution of risk analysis models should proceed with increasing heuristic value as the primary selection factor.

General Needs of Ecological Effects Analysis

Analyses needed for ecological effects assessment are more complex than those for exposure assessment. For exposure assessment, the spatial and temporal concentration of the toxicant is the most complex demand that is made of the analysis. Frequently, only a steady-state level is required. In any case, the interest is in the level of the chemical, a single entity. If

daughter products are of concern, they can be treated conceptually as separate, but linked, entities.

For ecological effects assessment, in contrast, the dimensions of the problem expand dramatically. Possible results of interest include lethality, any of the nonlethal effects, direct effects on any of the very large number of biotic species present, and any indirect effect brought about by alteration of characteristics of the interacting biotic species. In the presence of this wide variety of effects at different hierarchial levels of organization, it is understandable why the particular effects of interest for regulatory needs have not been made explicit. If any of these kinds of effects were known to be very likely to occur as a result of the environmental introduction of a chemical, undoubtedly that information would be used as strong evidence for regulating the chemical.

Ideally, therefore, a model for analysis of ecological effects would possess the capability to predict responses at all these levels. It is widely recognized that we are quite far from the state of knowledge required to produce a model with all these capabilities.

Nature of Ecological Risk Analysis

The intent of ecological risk analysis is to determine the nature (qualitative description), the extent (quantitative description), and the probability of the effects expected to occur in ecosystems as a result of the introduction of toxic chemicals. Because effects of many sorts can occur to many kinds of organisms, and because indirect effects such as alteration of environmental chemical concentrations can be induced, the description of the nature of risk is likely to be very complex.

To be useful, every effect description needs to be accompanied by quantification of its magnitude, which could have spatial and temporal dimensions, and therefore, the statement of extent will be even more complex. A single risk assessment could yield a set of information, which, if presented uncritically as a set of effects, extents, and probabilities, could be too complex to be useful. Multiplied by the number of environments that could be affected, the task of assimilating the information could be formidable indeed. It is readily apparent that an organized approach to risk analysis is necessary. The ideal approach would facilitate obtaining the greatest degree of comprehension of the expected behavior and effects of the chemical, using as few specific measurements on the chemical as possible.

Specific components of a predictive ecological effects model depend on the resolution of the model. As proposed here there would be three major levels of resolution:

- o steady-state analysis for chemical activity of toxicant in selected populations,
- o analysis of time-dependent chemical activity of toxicants in the body and the resulting effects in selected populations and small sets of interacting populations, and
- o ecosystem level of analysis in which direct effects are considered in the context of ecosystem interactions as well as indirect effects resulting from ecosystem interactions in the presence of direct effects.

Exposure analysis, as currently practiced, provides most of the needed information on environmental distribution. The primary quantity that is not routinely obtained in exposure analyses is the probability of exposure. Such a probability statement ideally would give the probability of occurrence of

various concentrations. For some analyses the statement would give the probability of occurrence of concentrations as a function of time, e.g., where chemicals are introduced seasonally.

A steady-state analysis of body burden can be carried out without reference to specific organisms, provided that chemical activity in the source is known. This analysis can be conducted, accounting for body burden arising solely from transport (exchange between organism and environment, no metabolic transformation) that is driven by chemical activity gradient. In this type of analysis, the organism is represented as comprised of as many compartments as necessary to achieve the specificity of chemical activity coefficient to achieve the desired level of resolution of the exchange process. For example, for neutral hydrophobic chemicals, the analysis could be carried out using three compartments: water, lipid, and all other materials combined. Bioconcentration values, if desired for specific organisms, can be obtained by substitution of the appropriate value for fraction of whole body comprised of fat.

As we have formulated the steady-state analysis, organisms of all kinds achieve the same body chemical activity of toxicant, when all are exposed to the same source of chemical activity for long periods. Where conditions make this assumption inappropriate, this level of analysis should not be used alone. A well implemented model would provide a guide as to when results based on the steady state assumption might mislead the user. Measures of toxicity from laboratory tests can also be expressed in terms of chemical activity of the exposure concentration.

We assume that death occurs to any individual when the chemical activity in its body exceeds a critical level, the same level for all individuals. To consider lethality in the steady-state analysis, one could analyze only for

whether the lethality threshold would be exceeded for long exposure times. If quantitative dose-response relationships exist for nonlethal effects, however, one could also give the response level expected.

Body chemical activities exceeding or even approaching the toxic levels obviously signal high risk, but in addition, risks due to incremental loads can be inferred. Any body burden of a toxicant reduces the capacity of the organism to withstand additional exposures to that toxicant. The steady-state analysis of risk can be combined with the next level of complexity of analysis, the population dynamics level, to obtain risk to populations at some steady state with a toxicant source when transient loads are introduced in the presence of background load.

At the population dynamics level of complexity, temporal factors can be brought into the analysis. In this level of analysis, the temporal patterns of increase in body burden and of mortality are obtained. To analyze for the effect of neutral hydrophobic organic chemicals on specific populations, exchange rate and distribution of fraction fat are items needed in addition to those used in the steady-state analysis. Without significant additional complexity, exploration of effects of toxicant on population interactions can be conducted. For example, the dynamics of a fishery population, whose population parameters are well known, could be explored for direct effects of toxicant and for effects when both prey population and the fish population are exposed to the toxicant. As noted, this level of analysis can be combined with the steady-state analysis to obtain the expected transient response of populations at equilibrium with an environmental source when transient loads are introduced in addition to the background levels.

At the most complex level of analysis, ecosystem effects can be explored. Consideration of effects at the ecosystem level is not a simple expansion of

analyses of population interactions. In my view, ecosystems differ from arbitrarily selected biological associations of interacting organisms, not merely in the number of members or the degree of interaction among them. The completeness of the kinds of processes and interactions that occur is the distinguishing difference.

To perpetuate themselves, organisms must carry out reactions that yield energy and that build biomass. A model of a biological association of a group of interacting organisms would represent energy metabolism and biosynthesis in one or a few ways; a model of an ecosystem would represent them in as nearly complete a manner as knowledge and practicality permit. Ecosystem effects would be any effect whatever, such as effects on any of the processes of energy metabolism or biosynthesis, including those types of effects of interest in the lower level analyses. Of course ecosystem effects would include those effects observable only in ecosystems such as alteration in levels of environmental chemicals or total power output. Model analysis for effects would be carried out using difference in affected and unaffected performance of any observable quantity of interest, appropriately normalized.

There are two major benefits of the ecosystem level of analysis beyond those that are achievable in the lower levels of analysis. The most obvious is that effects on the interactions that are represented only in the ecosystem model can be predicted only via the ecosystem model. The second is the possibility of examining the implications of specific effects, the "so what" question. Effects that are observed often are not, themselves, of concern. Presently we have a very restricted ability to predict indirect and long term consequences that are expected from an observed effect. At the ecosystem level, analyses can be conducted to determine the long-term consequences of an exposure. A capability of analyzing for indirect and long-term consequences would be a major advance over present capability.

Using all three levels of complexity is one aspect of organizing a risk analysis so that the information can be assimilated. An efficient means of selecting environmental conditions to be represented in an analysis would also contribute to organizing the analysis. Considering a range of conditions spanning all environments that the chemical is likely to enter might be possible for a steady-state analysis, and if a coarse grid is used to represent the environmental characteristics, for a population-dynamics analysis.

Although no specific implementation exists for a comprehensive ecosystem analysis, one cannot readily visualize routine analyses over a wide range of conditions with an ecological system model, because of the probable time requirements. The need for probabilistic estimates further complicates and increases the computational time of ecosystem models, because the only general approach for generating probabilities for such complex models is the Monte Carlo technique. How best to use this level of analysis covering relevant environmental conditions and biotic systems efficiently probably will be determined only after the models have been applied. It is possible, however, that preselection of conditions and biotic systems can be accomplished as part of the model development, so that, in practice, selective use of a few scenarios can be used heuristically to reduce the need to one or a very few additional analyses.

Proposed Toxicological Models

The three levels of resolution of the analysis differ in temporal resolution and in the comprehensiveness of coverage of the ecosystem. They do not differ in the assumptions made for the toxicological models. The toxicological model proposed expresses mortality as a function of exposure to a chemical and is based on simple assumptions.

The specifics of this model arose through interaction of a group at EPA's Athens Environmental Research Laboratory. We assume that toxicity is expressed as a result of internal chemical activity as reviewed by Filov, et al. (3). Most toxicity data are expressed in terms of the concentrations to which the organisms were exposed. Interpretation of these data to infer internal concentrations would require an exchange model. Although chemical exchange in general will be a function of the chemical activity gradient, charged and polar chemicals often are handled differently by biological membranes via active transport or other mechanisms. Therefore separate models for exchange will probably be needed for these categories of chemicals.

The toxicological model for lethality consists of three major components: chemical exchange, a threshold level of chemical activity, and a capacitance of the organism's body for the chemical due to fat content. We assume that velocity of exchange is in proportion to the internal to external gradient of chemical activity. Finally, we assume that death occurs in a population when individuals reach a critical chemical activity in blood or other effect compartment and that this quantity is a population parameter with very low variance. Distribution of time to death in a population (or equivalently, of fraction dead after a time interval) is a function of the distribution of fat in the population, because the greater the fraction of an organism comprised of fat, the greater is the quantity of chemical that must be absorbed to reach a given body chemical activity, e.g., the threshold lethal chemical activity.

The composition of blood is relatively constant, so one would expect chemical concentration in the blood to be about as good an indicator of toxicity as chemical activity. In fact, Mount and coworkers (4) found evidence for a

lethal threshold level of endrin in the blood of catfish in an experiment in which a range of lethal and nonlethal exposure concentrations was applied. Brungs and Mount also reported values of lethal blood concentration of endrin in gizzard shad experimentally exposed and collected in fish kills (5). These values support the hypothesis of a threshold lethal blood level.

Making these assumptions leads to a model for chemical exchange that produces bioconcentration values consistent with usual observations. Specifically, bioconcentration referenced to fraction of the body comprised of fat typically varies much less than does the same data set referenced to whole body. Use of an appropriate value for lethal activity (or lethal aqueous blood concentration) together with a descriptive statistical distribution for the distribution of fraction fat in the exposed population permits calculation of the probability of death as a function of time, or calculation of fraction dead after a fixed exposure duration. In this approach, fraction fat is the only random variable. Undoubtedly other factors vary, but we propose this as a simple hypothesis that can be easily checked by experiments and extended as warranted.

Toxicity models for nonlethal effects are yet to be fully defined for this effects model. Evidence indicates, however, that an approach similar to that proposed for lethal effects will be fruitful for nonlethal effects as well. There are at least two complicating factors. First, mortality is a more precisely identifiable endpoint than are many of the nonlethal effects, and as a result, greater observational error is expected when studying nonlethal effects. Second, many of the nonlethal effects will be quantitative responses in contrast to lethality, which is a discrete response. Thus, instead of a discrete lethal body chemical activity, we would seek to represent the nonlethal response by a dose-response function that describes individual response.

For both major components of the ecological effects models (the ecological component and the toxicological component), we take a strong biophysical point of view as our first major organizing principle. In this approach, we recognize first the existence of tendencies for chemicals to move and react in characteristic ways that are expressed in commonly measured properties. We further recognize that these tendencies exist equally in biotic and abiotic systems.

Our knowledge of these tendencies is poor for the interaction of xenobiotic chemicals with biochemicals in vivo to produce toxicity, but we expect much biophysical behavior, such as transport into and within organisms, as well as variation in biochemical behavior among similar chemicals, to reflect properties of the chemical. Where it appears that the natural tendencies of chemicals are not reflected in biotic systems, there is a basis to invoke special biotic mechanisms. In all such cases, biochemical work is done to counteract the natural chemical tendencies. Our approach, therefore, is first to account for as much of the observed behavior as possible on biophysical grounds, i.e., based on properties of the chemical and the organism, and only when biophysical models are shown to be inadequate, to invoke models for active transport and other energy requiring processes.

Equally importantly, the elementary components of our models are phenomena and quantities that are below the level of resolution of results from laboratory bioassays or from effects expressed in natural systems such as chemical and biological properties and biological structures and capacities. This gives the possibility of representing both laboratory and field behavior on a common basis, and in fact, starting at this level is a necessary condition for making such predictions. Clearly, however, it is not a sufficient condition.

Sufficiency is much more difficult to achieve. The relevant specifics of environmental chemistry and toxicology must be organized and included in our models. The point here is that the biophysical approach provides the structure on which the necessary subject matter can be organized.

The Proposed Ecological Models and Their Scopes

The second major organizing principle for ecological effects modeling is to recognize that the scale of resolution of the model is established by the kinds of questions that are to be asked. It is in anticipation of the questions that are expected that the three levels of resolution already discussed have been selected. In keeping with these proposed three levels of resolution of the ecological effects model, three scopes of representation of ecological components are needed.

Using the steady-state analysis to compute chemical activity of toxicant in organisms, we can speak generally of aquatic organisms without reference to particular species. It is feasible, however, to build steady-state models of ecosystems representing each ecological role by a single state variable (6). If quantitative dose response relationships exist that can be applied generally, they could be used to infer altered steady-state ecosystem structure. These models would be highly generic and useful only heuristically.

The population level of analysis will be the most useful for questions that arise naturally from our current point of view. Current concern for effects of chemicals is for population effects, particularly for rare or economically important species. It is feasible to permit introduction of a high level of physiological and behavioral detail into the population equations, and thereby achieve a high degree of specificity.

Such characteristics as reproductive periodicity, specific rate of increase, age structure, age-specific natural mortality, feeding characteristics (plankton

feeder, ambush predator, pursuit predator, etc.), characteristics of food species, and other properties of the organism of interest can be incorporated in the population equations. It is unlikely, however, that effects on very many of these characteristics will be known. If the effect of a toxicant on general characteristics is known, such as alteration of metabolic rate, alteration of sustained activity capacity, or of maximal swimming rate, etc., then alteration of many of the activities can be inferred. Use of a model structured so that many of the possible behavioral and physiological effects can be explored for their implications for the population maintenance could identify useful endpoints that are not being utilized currently.

To explore the response of a single population to exposure to a chemical, one would establish a particular pattern of all extrinsic influencing factors that are considered by the model. Often this pattern would simply be constant values for all factors, but a well implemented program would allow seasonal or other important temporal variations of the intensity of influencing factors. At this same level of resolution, it is feasible to permit a very few of the factors to be state variables, e.g., food species whose population densities might be under partial control by a predator species of interest. Thereby, the effects of a toxicant on small interacting groups could be explored.

To explore effects on higher levels of interactions and resulting environmental properties, a model of an ecosystem is the appropriate structure. Questions that relate appropriately to this level of resolution refer to the possibility that other effects occur besides those detectable by studies on populations of single species or of very small sets. One question that would follow immediately upon completion of analyses at the population level of resolution is whether effects detected at that level are reproduced in the

context of a complete ecosystem and, further, whether these effects cause other effects to cascade through the system.

Would such a cascade of effects, beginning with one that is expressed acutely at one point in an ecosystem be damped in a multispecies representation of an ecosystem? Are recovery time constants obtainable from ecosystem models? Perhaps the most important kind of question concerns ultimate effects. Many effects have been identified and suspected of indicating deleterious effects in ecosystems, but have not been taken seriously because it has not been possible to infer that a serious consequence is likely. A theoretically sound and well implemented ecosystem model would provide for this kind of analysis. Considerable research is needed with ecosystem models to achieve the capability to implement such a model.

There are many points of view among ecologists as to the appropriate components of an ecosystem model. The appropriate components, however, cannot be identified apart from the questions to be asked of the model. If our prognosis of the nature of the questions is approximately correct, many of the questions will be about system level effects and possible cascades of effects when exposure or other effects are known to occur to specific populations of higher organisms. Other questions will concern environmental chemicals, the levels of which are under partial control by the inputs and outputs of a large number of microbial populations. It is apparent that components should include environmental chemicals, microbial populations, and populations of higher organisms, coupled in such a manner and described at a resolution such that both direct effects to individual components can be expressed, and also indirect effects one or more steps removed from the direct effect.

To begin to implement such a model, it is useful to seek an organizational scheme. Such a scheme has been discussed (7) and involves categorizing organisms based on their source of energy and their source of materials for biosynthesis. This categorization results in consideration of three major trophic types -- photoautotrophy, chemoautotrophy, and heterotrophy. Heterotrophy is divided into the higher organisms and microheterotrophs, which are further subdivided by the oxidant (terminal electron acceptor) used. Models are worked out to represent the dynamics of each type based on thermodynamic yields from the energy equation and the cost of biosynthesis and metabolic activity for maintenance and other activities of each trophic type (7). These equations are expressed in the detail that would be useful in the population level of analysis, so implementation of an ecosystem model would entail simplification of some of the expressions.

This organizational scheme provides more than just a categorization of the kinds of organisms to be represented in an ecosystem model. The categories are sufficient to permit representation of complete element cycles by the model. By complete element cycles, it is meant that a set of biologically mediated redox processes that are sufficient to transform elements among the major chemical forms are represented.

Representation of complete element cycles makes it possible to consider the effects of a toxicant on any of the major element cycling processes. It also places a realistic constraint on the behavior of the ecosystem model by bringing chemical concentrations into the model as state variables rather than specifying their level via boundary conditions. Quantities and concentrations of imported environmental chemicals would be specified by boundary conditions.

Representing all the categories of organisms required to carry out the complete set of redox reactions represented in the model requires that a minimal

level of spatial heterogeneity also be represented. Specifically, it will be necessary to represent environments that may be depleted of O_2, NO_3^{-1}, and other oxidants, if elements are to be transformed from oxidized chemical forms to reduced forms. Furthermore, because transport of chemicals between oxidizing and reducing environments is an important process in element cycling and general system functioning, it will be important to pay close attention to representing spatial dimensions and the nature of interfaces between different environments as well.

Measurable Endpoints for Environmental Risk Assessment

Development and use of ecological effects models can be interactive with the processes of identification and measurement of endpoints for environmental risk assessment in two ways. First, where possible, ecological effects models should be expressed in terms of presently measured quantities. Quite obviously, doing so makes it possible to utilize data that have been obtained for use in environmental protection. As an example, in this work it was desirable to express the toxicological model in terms that would permit use of laboratory bioassay data.

One cannot expect to directly and theoretically relate predictions of expected effects in natural systems to effects as measured by bioassay procedures, because both concepts are at the same level of resolution and integration. Our approach has been to predict both on a common theoretical basis using lower level concepts as already discussed. If fully successful, this approach could provide a greater applicability to laboratory bioassay data, as well as provide a rationale for useful modifications and additional measurements.

Development and use of ecological effects models can help to identify quantities that might be useful as regulatory endpoints. We would expect these to show up in two ways. First, it may be found impossible to express models in ways that utilize currently measured endpoints, so that additional quantities are needed to permit analysis. Second, if models are used routinely, it is expected that certain quantities are repeatedly identified as indicating environmental damage that should be prevented through regulation. These or related quantities would be candidates for endpoints that should be measured in environments during monitoring, or in test systems during preregulatory testing.

Desirable Attributes of a System Implementation

The primary purpose of an ecological effects model is to serve as an evaluation aid in the regulatory activities of the Office of Pesticides and Toxic Substances in the United States Environmental Protection Agency. Its implementation, therefore, must directly reflect their needs. Experience with EXAMS, however, has shown that a very large interest exists in other government agencies, state governments, industry, and in academia. We anticipate that a similar interest would exist in a well implemented ecological effects model. A well implemented model, in our view, would follow modern views on program design. It would be highly structured, modular, and therefore, extensible. It is necessary to follow this approach to program design because the field of ecotoxicology is rapidly changing and growing, and to satisfy its primary purpose, the model would change to reflect this growth. The initial design must accommodate frequent alteration without necessitating complete code revision.

The staged approach to analysis: steady state, population, and ecosystem levels, will place special demands on design of the input and output interface

with the user, and will benefit from a structured approach to program design and implementation. Not all aspects of the input-output interface will be implemented simultaneously, and this will necessitate modularity of program code, a characteristic more readily achieved using structured methods of program design and implementation. To provide a sense of continuity, there should be a high degree of similarity in the input and output interfaces for the different levels of resolution of analysis. The input interfaces will be used to define and control the analysis, and the output interfaces will be used to examine the output files.

The similarity among the levels of analysis should include syntax and vocabulary with nouns and modifiers selecting and establishing the level and scope of analysis. In response to a query, the presentation of results should be qualitatively similar. All these concepts and characteristics are achievable in a system implemented primarily at the personal computer level (depending on the method selected to implement the ecosystem level of analysis, a mainframe might be invoked for computationally demanding portions of the system), so the code should be highly portable and highly usable. Prospects for an ecological risk analysis modeling system, therefore, are judged to be good.

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